A Dissertation on

A CLINICAL STUDY ON TEMPORAL PALLOR OF THE OPTIC DISC

Submitted to

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CERTIFICATE

This is to certify that **Dr.Thirumangai.S.,** Post Graduate student in M.S Ophthalmology, at Regional Institute of Ophthalmology and Government Ophthalmic hospital attached to Madras Medical College, Chennai, carried out this dissertation on **"A CLINICAL STUDY ON TEMPORAL PALLOR OF THE OPTIC DISC"** under my direct guidance and supervision during the period from May 2006 to March 2009.

This dissertation is submitted to the TamilNadu Dr.MGR Medical University, Chennai in partial fulfillment of award of M.S. Degree in Ophthalmology

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I, **Dr.Thirumangai.S.,** solemnly declare that the dissertation titled **"A CLINICAL STUDY ON TEMPORAL PALLOR OF THE OPTIC DISC"** has been prepared by me. This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of M.S. Ophthalmology, degree Examination to be held in March 2009.

Place: Chennai

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CONTENTS

PART I

INTRODUCTION

Temporal pallor of the disc is a very frequent fundus finding in a number of patients attending Eye hospitals. This has to be differentiated from the relative pallor of the temporal part of disc in normal people. A variety of disease processes present with temporal pallor . The etiological cause of this entity is of diagnostic challenge.

In this study , the criteria for etiological diagnosis , the exact pathology , the various causes , clinical features, management and their course and prognosis are discussed.

II Review of literature

2.1 Anatomy of the visual pathway

The visual system as a whole is developmentally, morphologically and functionally an outgrowth from the central nervous system. The visual sensory system comprises of retina, optic nerve, optic chiasma, optic tract, lateral geniculate bodies, optic radiation and the visual cortex.

The rods and cones of the neural epithelium of the retina form the end organ of the visual afferent tracts. The first conducting nerve cell of the first order is the bipolar cell of the inner nuclear layer of the retina with its axon in the inner reticular layer. The neurons of the second order are the ganglion cells in the retina, the processes of which pass into the nerve fiber layer and along the optic nerve to the lateral geniculate body. The optic nerve contains approximately 1.2 million nerve fibers and is divided into intraocular, intraorbital, intra canalicular and intracranial till it reaches the optic chiasma.

In general, it may be said that the fibers from the peripheral parts enter the periphery of the optic nerve, while those from parts of retina near the optic disc enter the centre area of the nerve. The fibers from the macular region enter the nerve on its outer aspect in a triangular area with the apex

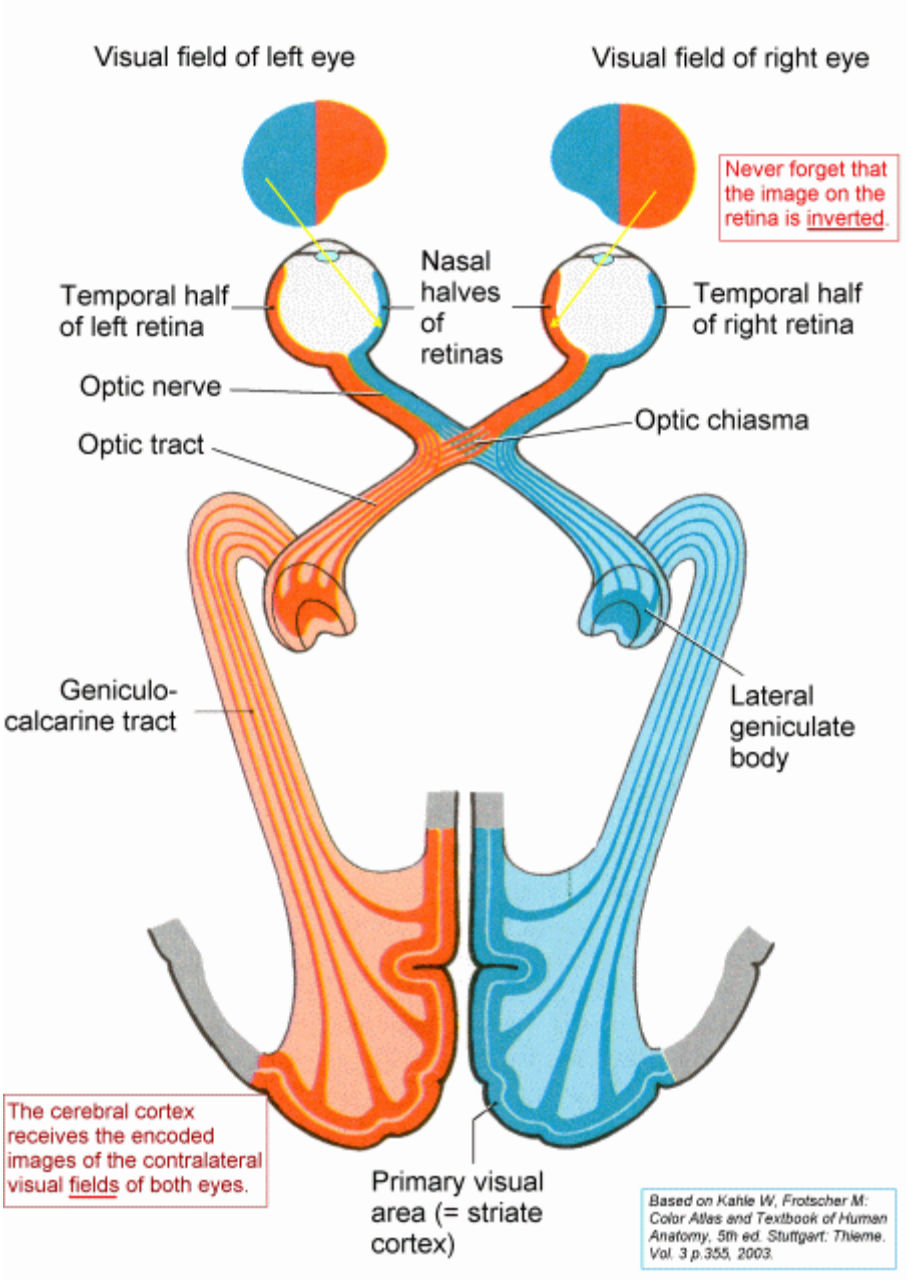


Figure 1-VISUAL PATHWAY

towards the cortex.This is called papillomacular bundle which become centrally situated in the posterior part of the optic nerve. The retina is divided into nasal and temporal halves by an imaginary vertical line passing through the fovea . Fibers from the temporal half of retina enter the chiasma and pass into the optic tract of the same side and synapse in layers 2,3,5 of the LGB. Fibres from nasal half of retina enter the chiasma, decussate and pass into the optic tract of the opposite side and synapse in layers 1,4,6 of LGB. Each LGB, thereby receives visual information from both retinas. The corresponding third order neurons originate in the LGB and then pass by the optic radiations to the corresponding occipital lobes. Lesions of the optic tract or occipital cortex will cause blindness of ipsilateral temporal retina and contralateral nasal retina (Hemianopia).

The visual fibers in the optic radiations, run behind the motor fibers in the internal capsule .Thereafter they separate considerably, the lower fibers of retina run forwards into the temporal lobe (Meyer's loop) before they turn backwards to the lower portion of visual cortex. The dorsal fibers run backwards in a more direct course to the upper part of the visual cortex. They pass close to the posterior cornu of the lateral ventricle, so that when the ventricle is distended they may be subjected to pressure here.

The occipital cortex in and about the calcarine fissure differs from the cortex elsewhere in the possession of the white line, the line of Gennari. This primary visual sensory area is the cortical projection of the corresponding halves of both retinae. The parts above and below the calcarine fissure represent the upper and lower corresponding quadrants of both retinae respectively and the posterior part of the occipital lobe represents the macula.

2. 2 Normal appearance of the disc:

Physiologic temporal pallor :

An absolute prerequisite to recognition of the abnormal disc is familiarity with normal disc colour. The temporal side of the normal disc usually is paler than nasal side. This may be due to the following causes:

i) The size of the physiologic cup – when it extends almost to the temporal edge of the disc, the temporal side looks pale.

ii) Thin translucent nature of the temporal nerve fibre

iii) Relative sparseness of capillaries on that side of disc

iv) Sometimes a temporal crescent of sclera exposed by retraction of RPE may enhance whitish appearance of disc.

v) In eyes with axial myopia, the normal temporal pallor is accentuated because of the oblique entry of optic nerve, the contents of the disc are displaced nasally. The physiologic cup is shallow and its extension to temporal margin produces relative temporal pallor.

vi) In infants, efforts to keep lids separated while doing fundus examination, may produce inadvertent pressure on the eye and may account for apparent temporal pallor.

vii) Other variable factors like the amount of light used, the colour ,temperature of the light source and the age of lens may give an illusionary picture of whiteness of disc.

2.3) Acquired or pathologic temporal pallor of the optic disc

When the optic nerve degenerates, its blood supply is reduced and the smaller vessels are no longer visible. In addition to reduction in blood supply, formation of glial tissue has been said to occur with optic atrophy. These two factors have been presumed to account for the pallor that is associated with optic nerve atrophy.

Pallor of the optic disc may be diffuse or confined to one sector. Kestenbaum introduced a “Capillary number test” is which the small vessels at the margins of the disc which are usually 9-10 in number were decreased in patients with diffuse optic atrophy.

Acquired temporal pallor is most commonly associated with segmental optic atrophy. The white area which generally extends from the temporal edge of disc to the central vessels, may appear totally devoid of capillaries. Margins of this white area tend to blend gradually with the reddish yellow colour of the surrounding disc tissue. Sharply demarcated wedge shaped temporal pallor is the consequence of discrete papillomacular bundle lesions that occur in the retina between the macula and the disc.

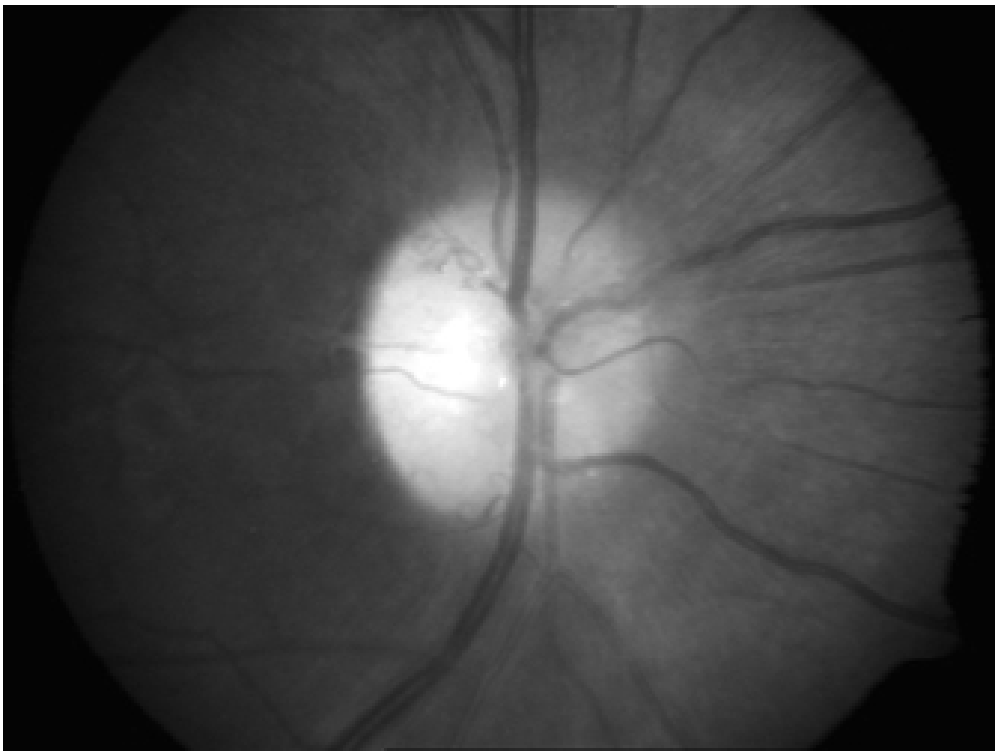


Figure 2-TEMPORAL PALLOR OF THE OPTIC NERVE HEAD

Superior, inferior or nasal sector shaped pallor seldom appears as sharply circumscribed as temporal pallor.

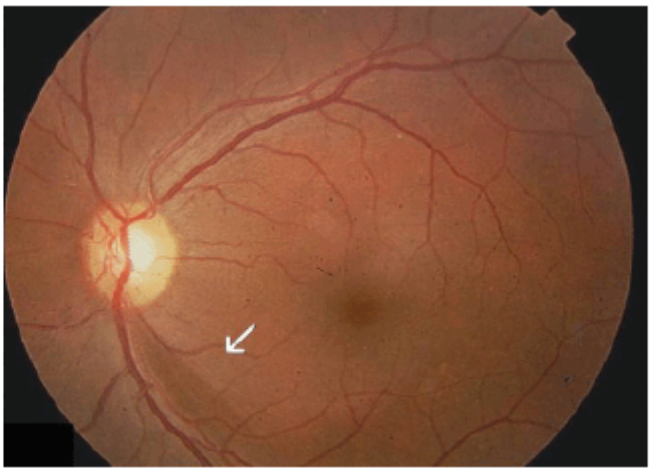
Acquired temporal pallor is usually caused by optic neuropathies that selectivity affect central vision and field, sparing the peripheral field. Such optic neuropathies include toxic and nutritional optic neuropathies, dominant hereditary optic atrophy and optic neuritis. when superior or inferior disc pallor is present, an ischaemic etiology is more likely.

The evaluation of optic disc color, though looks simple has many pitfalls. More objective evaluation of the pale disc can be obtained by detailed observation of its configuration and neural tissues, its veins, arteries and capillaries and the peripapillary retinal fiber layer that surrounds it.

Evaluation of peripapillary retinal nerve fiber layer in the diagnosis of optic atrophy

Focal destruction of nerve fiber bundles is one of the common pathologic denominators of disease that affects the inner retinal layers, optic disc, retrobulbar optic nerve or a combination of these structures. When bright red free light is used in ophthalmoscopy or in fundus camera, normal linear striations that overlie the retinal vessels are seen which are the peripapillary retinal fiber layers.

Figure 3-Localised nerve fibre layer defect



Early focal loss of axons is represented by the development of dark slits or wedges in the peripapillary retinal nerve fiber layer. The slit defects are most easily identified in the superior and inferior arcuate regions where the nerve fiber is particularly thick. When multiple nerve fiber bundle defects are present, they impart a “raked appearance” to the nerve fiber layer. With gross loss of axons, the defects coalesce producing a large wedge pattern. Within the wedge, the entire retina takes on a flat granular appearance with no striations being appreciated. The vessels in this area, having lost their surrounding nerve fiber layer covering, appear darker than normal. A prominent light reflex usually emanates from the side of vessels. As per the zone affected, one can interpret the possibilities, for eg, in temporal pallor due to toxic optic neuropathy, the papillomacular bundle will show dark granular appearance without the usual linear striations. In diseases like multiple sclerosis, the typical nerve fiber bundle defect can be detected even prior to the disc pallor and aids in the detection of multisystem involvement.

Diffuse thinning of nerve fiber layer around the optic disc is difficult to recognize in the early stages. In more advanced stages, signs of atrophy include decreased opacity of arcuate nerve fiber bundles, enhanced linear

highlights on large and small retinal blood vessels, apparent reduced caliber of blood vessels and pallor of optic disc with decreased visibility of the disc capillaries(Figure 3)

Retinal vascular changes associated with optic atrophy:

In most cases of optic atrophy the retinal arteries are narrowed but not all cases are associated with retinal vascular changes. In eyes with optic atrophy from damage to the retro laminar optic nerve, the retinal vessels are often unaffected. Eyes with retinal vascular changes associated with optic atrophy presumably have suffered an additional insult directly affecting the retinal vasculature.

In addition to the fundus appearance, several tests like visual acuity, fields, color vision help in deciding the pathological nature of temporal pallor.

2.4) Pathology of Optic Atrophy:

Since the axons in the optic nerve arise almost entirely from the ganglion cells located in the retina, damage to such axons may occur at several locations.

a) The retinal nerve fiber layer or optic disc

b) From disease within or surrounding the different portions of the optic nerve

c) From intracranial diseases of the optic chiasma, optic tracts or LGB

d) From diseases of the retrogeniculate pathways that produce transsynaptic degeneration. Focal disruption anywhere along an axon causes degeneration of the entire axon and its cell body, the retinal ganglion cells. When large number of axons undergo such degeneration, gross shrinkage of the optic nerve occurs. i.e optic atrophy.

Figure 4-NORMAL NERVE FIBRE LAYER

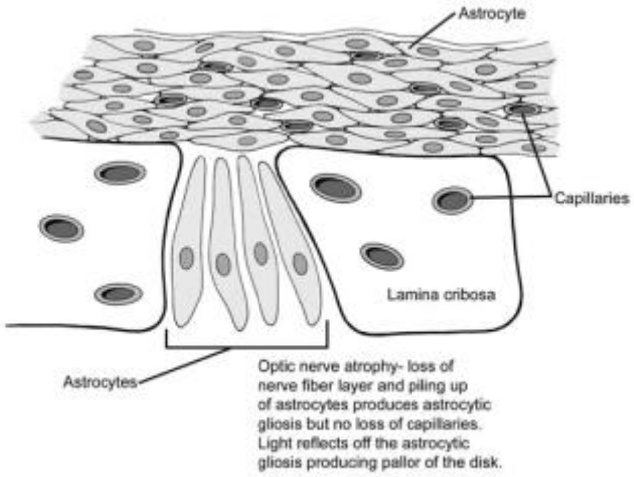
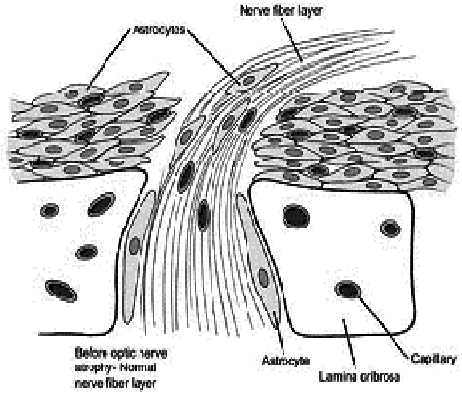


Figure 5-OPTIC NERVE ATROPHY



Wallerian ascending degeneration

When a visual axon is severed, its distal segment being separated from its nutrient ganglion cell body quickly disintegrates and disappears and its investing myelin sheaths undergoes a slower breakdown into simple lipids, this process is called Wallerian degeneration or Anterograde degeneration . The nerve fibres undergo wallerian degeneration at a rate inversely proportional to their thickness. Larger axons degenerate sooner and smaller ones latter. They break up into fragments including the myelin sheaths at constrictions. A series of ellipsoids form ,each surrounding a fragment of axons. Myelin is broken down into simpler lipids and engulfed by phagocytic microglia. Leucocytes are rarely present in Wallerian degeneration. An essential feature of Wallerian degeneration is the swelling and degeneration of terminal buttons of axons within the LGB. Wallerian degeneration and the optic pallor it produces can only be observed clinically when retinal axons are damaged within the eye .

Retrograde (Descending) Degeneration of Axons:

Although ascending degeneration begins and completes within 7 days, the proximal axon and the cell body remain normal for 3 – 4 weeks. During

this time, orthograde axonal transport continues in a normal manner. After about 3 – 4 weeks, the entire remaining structure degenerates rapidly, so that after 6 - 8 weeks, after severe optic nerve injury, no affected ganglion cells remain viable. A most fascinating feature of retrograde degeneration of optic nerve is that the time course of this degeneration is apparently independent of the distance of injury from the ganglion cell body.

Regeneration:

Regeneration of axon in the optic nerve is quite limited but some degree of remyelination does occur after injury. This has been proved in multiple sclerosis and other immune related disorders.

Remyelination plays a larger role in the recovery from acute and chronic compressive lesions of CNS as long as there has not been severe irreversible axonal dama

2.4.1 Pathogenesis of acquired disc pallor:

Optic atrophy is consistently associated with reduction in blood supply and formation of glial tissue. These two factors account for the disc pallor.

Experimental studies of animals suggested that factors that give the pink colour of the disc are its thickness and the cytoarchitecture of nerve fiber bundles passing between glial columns containing capillaries. They postulated that light entering the disc rim is normally conducted along the transparent nerve fiber bundles.The light diffuses among the adjacent columns of glial tissue and capillaries and acquire the pink color of the capillaries and the disc appears pink. In atrophic disc rim, the axonal bundles are destroyed and the remaining astrocytes are arranged at right angles to the entering light and little light passes into the disc substance to traverse the capillaries that are still present and surrounded by layers of astrocytes. Since light is reflected from opaque glial cells and does not pass through capillaries it remains white and the optic disc appears pale. In some areas, loss of tissue also allows light to pass directly to the opaque scleral lamina and this adds to the pallor. Other explanations for optic disc pallor include absence of capillaries and astroglial proliferation.

Discrepancies between the optic disc appearance and visual function:

No judgment of pallor is meaningful until and unless the pallor is correlated with optic nerve functions like visual acuity, contrast sensitivity,

color vision as well as quantitative perimetry, examination of pupils and electro physiological studies. An optic disc may appear pale but other examinations may not reveal any abnormality. In cases, it is most likely to be physiologic pallor. Conversely, an optic disc may appear normal despite optic nerve dysfunction. In most of these cases, careful evaluation of the disc, peripapillary retinal nerve fiber layer will provide evidence of retinal nerve fiber atrophy to produce pallor of disc.

2.6 **Etiology of Temporal Pallor** :

Several scenario could cause temporal pallor and the commonly encountered ones are

1) Nutritional and deficiency optic neuropathy

2) Toxic neuropathy including tobacco, alcohol and toxins

3) Retrobulbar, compressive and infiltrative optic neuropathy

4) Hereditary optic neuropathies like

    i) Leber's optic atrophy

    ii) Dominant optic atrophy

    iii) Hereditary optic atrophy with multisystem involved

iv) Sequelae of optic neuritis – demyelinating diseases

v) Chiasmal compression syndromes.

vi) Macular diseases

Since the causes are divergent , each entity in some detail is discussed in sequence.

2.6.1 Nutritional optic neuropathy

Toxic and nutritional optic neuropathies are often described together because they present with more less same symptoms.

Diagnostic criteria proposed by Lussel are :

1. Gradual bilateral painless progressive visual loss

2. Dyschromatopsia

3. Central or centrocoecal visual field defects

4. Optic disc appearance normal initially

5. Absence of metamorphopsia and hallucination

6. Improvement with treatment or removal of offending toxin.

Others :

1) Later developing temporal disc pallor

2) Atrophy of the papillomacular bundle layer

With the exception of vit B12, no specific nutrient has been proved to cause amblyopia in humans.

Vitamin B12 deficiency

This vitamin is found in milk, egg, meat and cheese and must be ingested. Vitamin B12 deficiency which takes years to develop generally arises in three different settings.

1) The first and most common is pernicious anaemia. This auto immune condition results in gastric atrophy, reduced intrinsic factor production by gastric parietal cells and B12 malabsorption. It affects middle aged individuals and can cause megaloblastic anaemia. An associated neurologic condition, sub acute combined degeneration of the spinal cord, bilateral optic neuropathy can develop. In this setting, patients develop both peripheral neuropathy and myelopathy. The second setting is in patients with a previous history of partial or complete removal of the stomach or ileum , rendering them unable to

absorb B12. The third setting, simple lack of B12 in the diet as might occur with a strict vegan is the rarest.

Clinical picture:

Patients present with slowly progressive vision loss with dyschromatopsia and centrocaecal scotomas. The optic nerve may be normal or may demonstrate pallor and nerve fiber defects . The defects could be temporal pallor, nerve fiber drop out in papillomacular bundle and rake like defects in the nerve fiber layer. Vision loss may precede the recognition of anaemia or other neurologic symptoms. Visual evoked response abnormalities have been found in patients without visual symptoms.

The diagnosis is confirmed by low serum B12 levels or elevated methyl malonic acid or homocystine , both of which require B12 for their metabolism.

Treatment is with parenteral hydroxy cobalamin and with prompt therapy, vision loss may be reversible.

Optic neuropathy has been shown in primate studies to be result of demyelination involving the papillomacular bundle. The pathogenesis remains unclear but may result from build up of toxic levels of cyanide

(especially in cigarette smokers) or improper fatty acid synthesis leading to myelin dysfunction. More recently Rizzo proposed that adenosine triphosphate deficiency may be a final common pathway for B12 optic neuropathy, leber's optic neuropathy and tobacco alcohol amblyopia.

Other vitamin deficiencies

Deficiencies in thiamine (B1), Pyridoxine (B6), folic acid, niacin and riboflavin (B2) have all been suggested causes of optic neuropathy

Thiamine

Deficiency of thiamine (B1) typically results in Beriberi or wernickes encephalopathy. There is experimental,biochemical and clinical data that suggest that thiamin deficiency may cause an optic neuropathy. However the experiments did not confirm conclusively that thiamine deficiency could be the sole cause of optic neuropathy. Ketogenic and high protein, low carbohydrate diets may also put a patient at risk for developing thiamine deficiency. Though there is good biochemical evidence that many of the patients with nutritional optic neuropathy were thiamine deficient, yet there is no basis to conclude that thiamine deficiency is the primary cause of an optic neuropathy.

In case of pyridoxine, there has been no causal relationship established between the deficiency state and optic neuropathy. However certain drugs like Isoniazid, cycloserine bind with vit B6.Other drugs interacting with B6 are chloramphenicol, hydrallazine, Pencillamine. It has been postulated that the optic neuropathy associated with these drugs are due to the pyridoxine deficiency rather than the drug themselves.

Niacin:

Deficiency of niacin causes pellagra, characterized by diarrhoea ,dermatitis and dementia. It may also have associated optic neuropathy characterized by central or centrocaecal scotoma , reduced visual acuity and temporal pallor of disc. Visual function improves if treated earlier.

Riboflavin vit B2

Riboflavin deficiency manifests less dramatically than Beriberi or pellagra. However retrobulbar optic neuropathy has been associated with burning feet syndrome among the malnourished prisoners of war in the far east which is specifically due to riboflavin deficiency.

Certain drugs like thallium may cause optic neuropathy indirectly by interfering with vit B2 metabolism.

Folic Acid

It plays a possible role in tobacco alcohol ambylopia.

Though there is no definitive evidence that such deficiencies play a primary etiological role in nutritional amblyopia, it remains possible that there are some patients who develop loss of vision from damage to optic nerves consequent to dietary vitamin deficiencies.

Nutritional amblyopia may be primary when it is due to poor dietary intake or secondary when it is associated with chronic alcoholism or smoking or malabsorption syndrome.

TROPICAL OPTIC NEUROPATHY:

There are optic neuropathies endemic to tropical regions for which a nutritional agent has been invoked. This epidemic was reported from Jamaica. The clinical features of the patients were numbness and cramps in hands and feet, bilateral visual loss, hearing loss, muscle wasting and pain, dermatopathy. In any case, no claim for nutritional etiology was made.

There is an otherwise unexplained bilateral optic neuropathy called “west Indian Amblyopia” The history is typically bilateral, painless, progressive visual decline in a well nourished adult. The field defects were central or centrocaecal scotomas but annular scotomas and peripheral constrictions have been reported. Optic atrophy develops. Deafness was also present. No treatment including various vitamin regimens is effective in reversing the visual or non visual manifestations of this disorder. Although a toxic basis (bush tea) has been postulated, these is no good evidence for either a toxic or nutritional etiology.A similar disorder has been described in Nigerians. The features include optic neuropathy, ataxia, peripheral neuropathy, hearing loss and an unusual feature was peripheral field constriction without central defects. The role of poor nutrition and ingestion of exogenous cyanide from an excess of cassava in diet was unclear. The evidence that cyanide plays a role in causing any optic neuropathy is discussed further in section on tobacco optic neuropathy.

2.6.2 TOBACCO-ALCOHOL AMBLYOPIA:

The most commonly recognized nutritional or toxic optic neuropathy may actually be a combination of both a nutritional deficiency state and a toxic effect from tobacco smoking.

The affected entity are heavy smokers, generally malnourished and they are usually pipe smokers or cigar smokers using shag and strong tobacco mixtures. Cigarette smokers are rarely affected Tobacco chewing and passive smoking in tobacco factories can also cause the disease.

Tobacco amblyopia is found mostly in middle aged or elderly males. The disorder is painless and characterized by slowly progressive bilateral dyschromatopsia and visual loss. The characteristic field defect is centrocaecal Scotoma . The optic discs are initially normal with pallor being a late feature. In alcohol amblyopia there may be central or centrocaecal scotoma especially for red with dense nuclei of scotoma in the centre and not in the centrocaecal area(Figure 6).

ETIOPATHOGENSIS

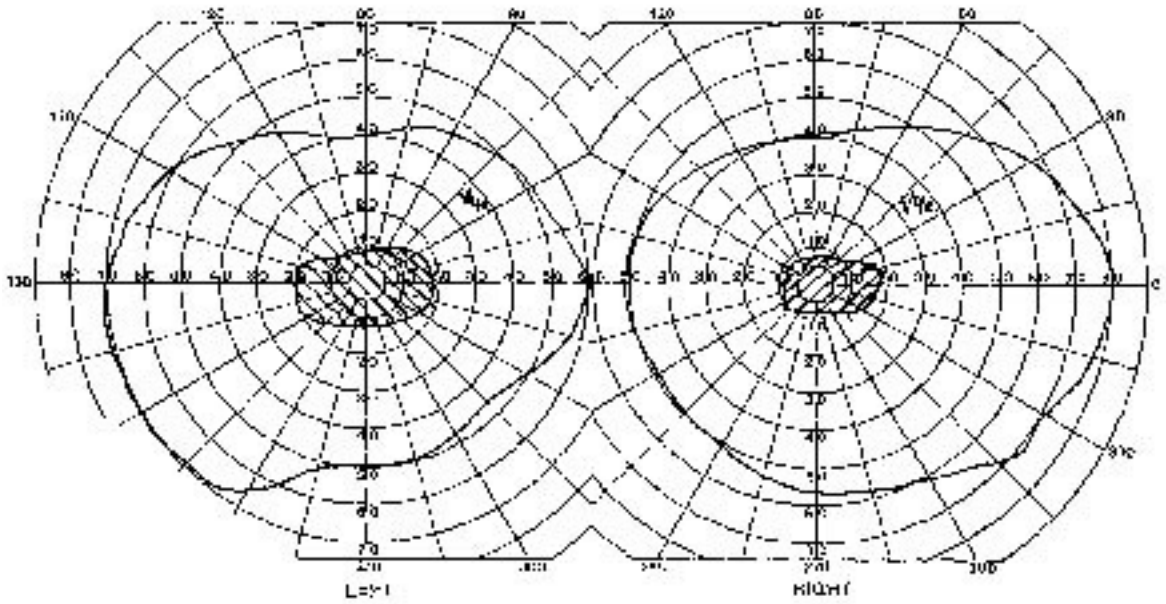
The mechanism by which tobacco damages the optic nerve has not been determined. One possibility relates to concurrent malnutrition. Vitamin B12 deficiency and impaired vit B12 absorption was found in 40% of patients. But in most cases of tobacco amblyopia, blood vit B12 levels were within normal limits.

ROLE OF CYANIDE:

Cyanide is present in tobacco smoke and this led to the suspicion that tobacco amblyopia is a limited form of cyanide poisoning. The cyanide theory was reviewed and concluded that there is a defect in sulfur metabolism and of cyanide detoxification in patients with visual loss associated with tobacco smoking. The role of cyanide in tobacco amblyopia is yet to be proved. As with other nutritional amblyopias, the etiology of tobacco amblyopia in many cases is multifactorial.

Patients with tobacco amblyopia slowly improve if they stop smoking or if they are treated with 1M injection of hydroxycobolamin 1000 micrograms, the dose being repeated five times at intervals of 4 days and then at 2 weekly intervals. Recovery may be monitored by VEP.

Figure 6-Centrocaecal scotoma of tobacco-alcohol amblyopia



Note :

Bilateral centrocaecal scotoma may at times mimic the bitemporal depression of chiasmal interference .The differentiating points are:

i) The VA is diminished

ii) The field defects extend across the vertical meridian .

iii) Especially demonstrable with red objects

iv) There is no peripheral hemianopic depression.

v) As the defects progress they appear more centrocaecal and less hemianopic.

Drug Induced retro bulbar toxic optic neuropathies

Insidious and slowly progressive bilateral loss of function in the central fields, with resultant diminished acuity, dyschromatopsia and central scotoma should alert the possibility of intrinsic optic nerve disease related to adverse effect of pharmaceuticals. Grant lists 40 such substances but the evidence for a statistical association between most substances and optic neuropathy is lacking.

1. Amantidine hydro chloride
2. Amiadarone
3. Arsenicals
4. Asperdium
5. Carbon disulfide
6. Carbon tetrachloride
7. Chloro dinitro benzene
8. Chlorpromazine
9. Chlorpropamide
10. Cobalt chloride
11. Digitalis
12. Dinitrotoluene
13. Disulfiram
14. Ethambutol
15. Ethylene glycol
16. Hexachlorophene
17. Hydroxy quinolines
18. INH
19. Lead
20. Lysol

21. Quinine
22. Streptomycin
23. Styrene
24. Thallium
25. Toluene
26. Trichloroethylene
27. Tricresyl phosphate
28. Vincristine
29. Amoproxan

Ethambutol:

It causes a dose related optic neuropathy. The toxicity is thought to result from its chelating properties. A typical presentation includes either central field defects with loss of acuity and dyschromatopsia or well preserved central visual function with peripheral field loss. Doses less than 15mg /kg/day are safe but in patients taking between 15-25mg/kg/day visual symptoms develop over a period of months. Dyschyromatopsia and loss of contrast sensitivity are the early signs. Some recovery usually occurs with discontinuation of the drug but abnormal usual fields and contrast sensitivity may persist.

INH:

It can also cause anterior and retrobulbar toxic optic neuropathy which can occur with or without pyridoxine deficiency induced sensory neuropathy.

AMIODARONE:

An optic neuropathy with disc swelling very similar to anterior ischaemic optic neuropathy has been reported in patients taking amiodarone. Visual loss may be insidious, with bilateral simultaneous involvement of eyes and is less severe than AION.

HALOGENATED HYDROXY QUINOLINES:

These drugs have been implicated in the development of myelopathy and optic neuropathy patients develop optic atrophy and severe vision loss that may be partially reversible with discontinuation of the drug.

2.6.3 RETROBULBAR COMPRESSIVE OPTIC NEUROPATHIES:

Compressive optic neuropathies can be identified by their typical presentation with slowly progressive visual loss and optic nerve atrophy.

SUMMARY OF THE IMPORTANT CAUSES OF OPTIC NERVE COMPRESSION:

INTRAORBITAL:

Primary orbital tumors

Cavernous hemangioma

Shwannoma

Secondary orbital tumors

Sinus tumor

Metastatic tumors

Enlarged extra ocular muscles

Intra orbital / Intracanalicular / Intracranial

Optic nerve neoplasmas (Primary)

Optic nerve glioma (Juvenile/benign)

- Malignant optic nerve glioma
- Optic nerve sheath meningioma

Optic nerve neoplasms(metastatic)

- Carcinomatous meningitis
- Optic nerve metastasis
- Lymphoma/leukemia/myeloma

Non tumour sinus causes

- Mucocele
- Paraclinoid tumours

Vascular causes

- Aneurysms
- Ectatic carotid arteries

Chiasmal lesions

- Pituitary adenoma
- Craniopharyngioma

Suprasellar meningioma

Other causes

Fibrous dysplasia, systemic diseases,thyroid disease,orbital hemorrhage.

Signs and symptoms:

Patients usually present with reduced visual acuity,colour vision and visual field defects typical of optic neuropathy.The optic nerve may be normal,pale and swollen.Compression will only result in optic nerve head swelling when the lesion is intraorbital and optic atrophy results in intracanalicular or intracranial lesions. The other symptoms are gaze evoked amaurosis, pain on eye movements, orbital fullness, proptosis and ptosis. The non ophthalmic manifestations are headache, facial numbness, anosmia, endocrine abnormalities.

2.6.4 HEREDITARY OPTIC NEUROPATHIES

There are 3 types of hereditary optic neuropathies.

1. Those that occur primarily without associated neurologic or systemic signs

2. Those that frequently have associated neurologic or systemic signs

3. Those in which the optic neuropathy is secondary to the overall disease process

Monosymptomaic Hereditary Optic Neuropathy

2.6.4 A LEBER'S OPTIC NEUROPATHY:

Since the disorder was described by Leber is 1871, the clinical profile of LHON has been well established, although it is highly variable in members of the same pedigree.

Age: Usually presents in second or third decade.

Sex: Men are affected more than women.

Mode of Inheritance: It became clear from studies that the disease was due to maternally inherited mitochondrial DNA.

CLINICAL FEATURES:

It causes an acute or sub acute failure of vision in one or both eyes.The average interval between first and second eye involvement is several months. There is painless vision loss, headache and uthoff's symptom may be associated. The patient presents with signs of an acute neuropathy, with central loss of vision & dyschromatopsia. Total blindness

is unusual. Field defects show central or centrocaecal scotoma. RAPD may be present.

DISC APPEARANCE:

Leber commented on tortuosity of vessels and white striated opacity in the peripapillary region .Findings also include

- Circumpapillary telangiectatic microangiopathy
- Pseudoedema (Swelling of nerve fiber layer around the disc)
- Absence of true edema or staining of the disc on FFA.

However, the disc may have a normal appearance is some cases. LHON patients subsequently develop optic atrophy. After the onset of optic atrophy, it is difficult to identify patients with LHON because pseudopapilloedema and telangiectasia usually resolve. Initially the nerve fiber loss is limited to the papillomacular bundle with temporal pallor of the disc. Later the entire nerve fiber layer disappears with diffuse disc atrophy.

Ophthalmoscopic changes appear to be present prior to any subjective or objective evidence of visual dysfunction and Farnsworth Munsell 100 Hue test appears to be the earliest indication of optic nerve dysfunction. Even asymptomatic family members of leber's disease have abnormal fundus

findings and whether all of these will develop the leber's disease is to be seen with time.

OTHER ASSOCIATED DISEASES :

Although rare, concomitant neurologic disease, cardiac conduction defects, arrthymias and skeletal deformities have been associated with LHON.

MITOCHONDRIAL GENETICS:

Although mitochondrial inheritance became the most plausible explanation, these mutations do not explain the variable occurrence of the disease within the same pedigree, and there was wide spectrum of phenotypic expression. Possible theories put forward for the variable phenotypic expression are 1) heteroplasmy (coexistence of mutant & normal mt DNA), 2) second genetic factor possibly on x chromosome 3) environmental factors like cyanide poisoning (cigarette smoking), alcohol and environmental toxins. These factors may possibly alter genetically programmed cell death.

TREATMENT:

There is no known effective treatment or prophylaxis for the visual loss that occurs with LHON. Steroids, cyanide antagonists, cyanacobalamine have been tried with variable results. Surgical dissection of thickened arachnoid tissue has been done in selective cases with disappointing results.

B. DOMINANT OPTIC ATROPHY

Two forms were described. Congenital or infantile form with nystagmus and juvenile form.

Patients typically present before the end of first decade of life with insidious onset of bilateral vision loss .They have central or centrocaecal defects with intact peripheral isopters. 40% of patients retain acuity of 20/60 or better. Visual loss usually does not progress once the patient reaches the second decade of life. Colour vision loss typically occurs along the blue–yellow or tritanopic axis. The associated optic disc pallor often takes on a striking wedge like appearance with temporal excavation of the neuroretinal rim, although the whole disc is sometimes pale. Because of the highly variable degree of visual dysfunction, seemingly asymptomatic adult and child relatives can often be identified if visual acuity or colour loss or optic atrophy is present. Recently pedigree analysis has identified the defect at the 3q21-3q28 chromosome. This has allowed for more accurate

identification of symptomatic and asymptomatic carriers. A much less common focus on 18q12.2-12.3 has also been identified.

PATHOLOGY:

Studies have demonstrated diffuse loss of retinal ganglion cells, suggesting primary ganglion cell death as the mechanism of disease.

TREATMENT:

There is no effective treatment at this time. Patients should also be screened for sensory neural hearing loss as this occurs with increased frequency in this group of patient.

C.RECESSIVE OPTIC ATROPHY:

The recessive form is more severe than the dominant form. Patient present in early childhood (age 2 to 4) with more profound visual acuity loss and searching nystagmus. There is often a history of parental consanguinity. There is usually diffuse disc pallor and attenuation of retinal vessels. Complicated, recessive optic atrophy when associated with spino cerebellar degeneration, cerebellar ataxia, pyramidal tract dysfunction and mental retardation is termed Behr syndrome.

OPTIC ATROPHY ASSOCIATED WITH NEUROLOGIC AND METASTATIC DISEASES;

Optic atrophy and progressive visual dysfunction frequently accompanies a variety of other inherited, degenerative neurologic conditions. Visual loss progresses along with the systemic neurologic syndrome but vision loss usually remains moderate. The disorders frequently involved are ataxias like spinocerebellar degenerations, Fredrich's ataxia, charcot maric tooth disease ,olivoponto cerebellar atrophy, Mucopolysaccharidoses, lysosomal storage disorders, Tay-sachs, Niemann pick A, krabbe's disease, Pelizaeus-Merizbacher disease, Adrenoleucodystrophy, Meta chromatic leukodystrophy, generalized gangliosidosis.

Progressive optic atrophy is also recognized in patients with Congenital deafness. In lysosomal storagedisorders,optic atrophy results from ganglion cell damage due to these accumulated materials.

2.6.5 TEMPORAL PALLOR AS SEQUELAE OF OPTIC AND RETROBULBAR NEURITIS:

<u>The causes of optic Neuritis:</u>

i) Local Conditions	**:**	Uveitis and Retinitis Meningitis Intra Orbital infections Nasal sinusitis, Venoms
ii) Demyelinating disease	**:**	Multiple sclerosis, Acute disseminated encephalomyelitis Myelitis, Devic's neuro myelitis optica , diffuse periaxial encephalitis
iii) Infective encephalitis	**:**	Viral, bacterial, rickettsial, Protozoal
iv) Systemic Conditions	:	Diabetes, anaemia, lymphomatous disease , collagen disease , endocrine disturbance , malignant disease , Toxic and allergic conditions, avitaminosis.
v) Giant cell arteritis		
vi) Neurological syndromes	**:**	Cranial polyneuritis Opticociliary neuritis

vii) Specific infections : Tuberculosis, Syphilis, mycoses

Clinical features of active optic Neuritis:

The vision loss is rapid in onset, progressive involving central vision more than peripheral. The loss may be mild to no PL. Reduced color vision invariably accompanies the vision loss .Red objects may be described as either pinker or browner in patients with dyschromatopsia , characteristic pain precedes the vision loss. Globe tenderness on pain on eye movements is typical. This pain characteristically disappears after 3-5 days. Patient complains of phosphenes or flashes of light. The presence of phosphenes should also raise the possibility of concomitant retinal disease as in neuro retinitis .Pupillary constrictions to light is illsustained and there is afferent pupillary defect on swinging flash light test. The characteristic field defect is central scotoma especially for red. Other possible defects are paracentral, arcuate, altitudinal and contraction of peripheral fields. Fundus examination shows nothing in case of retro bulbar neuritis but in papillitis there are disc edema, flame shaped haemorrhages, exudates, sheathing, cells in posterior vitreous. Though the disease is mostly unilateral (71%) it is bilateral and with simultaneous onset in 7% or bilateral but not simultaneous

but occurring with an interval of 3 months and less (12%) or more than three months.

The sequelae of optic Neuritis:

Vision: Visual acuity returns to normal or near normal mostly. In spite of the recovery of visual acuity to even 6/6 ,patients still feel that the quality of vision is not as good as the fellow eye and describe their vision in the affected eye as fuzzy.

Even after recovery people may experience uthoff's phenomenon i.e., blurring of vision with exercise, hot bath or emotional stress. This is common in patients with multiple sclerosis but also noted in optic neuritis of other origin as well as in leber's optic atrophy .

The possible explanations are:

1) Axonal conduction delay in demyelinating disease with heat.
2) Humoral substances released during exercise or heat causing a conduction delay.

Pulfrich phenomenon: Due to conduction delay ,simple to and fro movements of a ball on a string in one plane appears as elliptical movements. Due to this patient may have difficulty in driving in subway

and especially in parking. This is to some extent alleviated when the normal eye is given sunglasses so that some filtering effect occurs, neutralizing this effect.

Color vision: There is always some defects in color vision for (Red)which can either be easily made out even by the gross pseudo isochromatic charts or only by 100 Hue test in subtle defects.

Brightness sensitivity: Is reduced on the side affected by optic neuritis previously.

Pupil: Afferent pupillary defect is present in gross lesions. In subtle defects, the best method of detecting the defect is by edge pupil cycle time. In this method patient in seated before slit lamp and beam of moderate intensity of 0.5 mm thick horizontally is shone perpendicular to the iris to edge of the pupil; pupil constricts immediately and then dilates again, constricts and dilates. These oscillations occur indefinitely as long as the light is kept on. Now the time taken for 100 oscillations is counted with a stop watch and the time taken for a single oscillation is detected. This is called pupil cycle time (normal is 720-940 milliseconds. Normal difference is cycle time between 2 eyes 0-70millisecond). This time is significantly prolonged in a patient who has suffered optic neuritis. It is similar to VER

latency time in that it can detect and quantitate subclinical defects in optic nerve conduction time. It is an objective and quantitative method by which each eye can be tested individually. It is a fast, simple and reliable clinical test of optic nerve function.

Fundus examination: 50-80% of patients with optic neuritis develop some degree of disc pallor, whether initially the disc was normal or with swelling of the disc. The pallor is typically temporal but may also be generalized in some cases. Nerve fiber layer defects occur invariably either of papillomacular bundle or arcuate bundle or diffusely.

Visually evoked responses: Typically shows increase in the latent period but the amplitude may be normal or slightly reduced.

2.6.6 CHIASMAL COMPRESSION AND TEMPORAL PALLOR.

As a rule pressure in the chiasmal region produce bilateral simple optic atrophy with white optic discs with sharp margins. Involvement of chiasma is suggested by 1)monocular or bitemporal hemianopia,2)visual

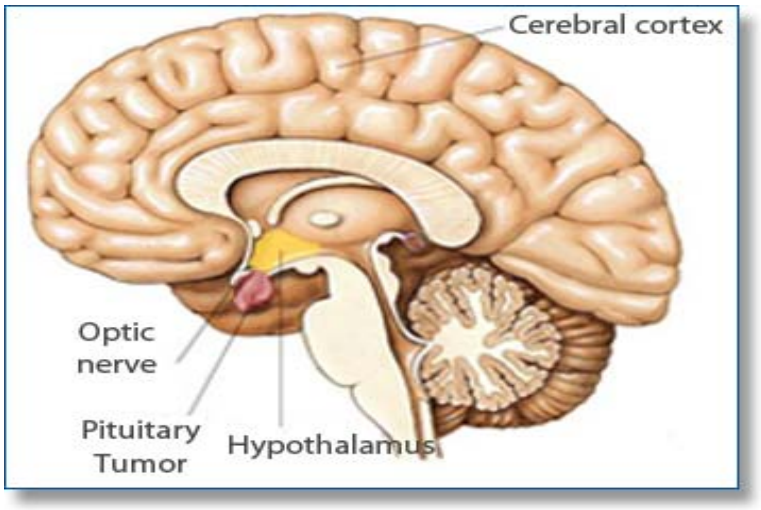
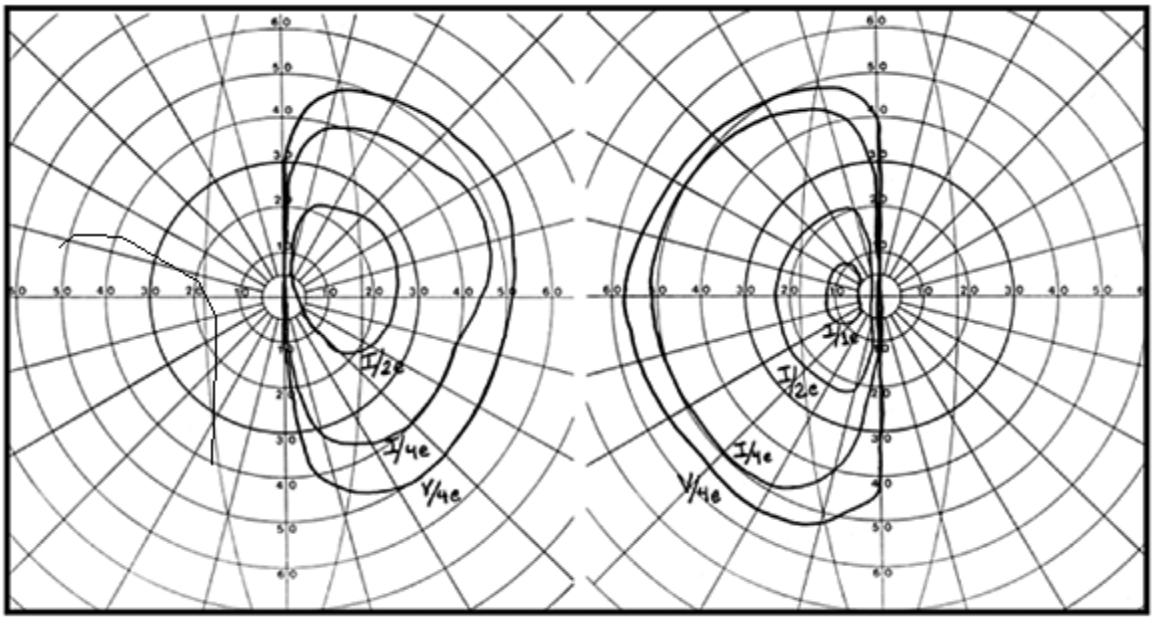


Figure 7-PITUITARY TUMOUR

Figure 8-BITEMPORAL HEMIANOPIA OF PITUITARY TUMOUR



loss of any type associated with endocrine dysfunction.In cases of bitemporal hemianopia, the pallor is rarely accentuated in the nasal half as all the macular fibres as well as those supplying the area between the disc and the macula pass through the temporal part of disc .So initially the pallor may be restricted to the temporal part of the discs alone but as the compression is unrelieved, the entire disc becomes pale. The most common causes are compressive sellar masses and therefore the diagnosis and management heavily depends on neuroimaging . This lesions produce visual acuity and field defects by interfering with the optic nerves, chiasma , optic tract or by obstructing the third ventricle and causing chronic atrophic papilloedema . So an awareness of temporal pallor which may occur due to chiasmal compression may aid in the early diagnosis of tumors compressing chiasma(Figure 7&8) .

2.6.7 MACULAR DISEASES CAUSING TEMPORAL PALLOR.

If carefully examined, the vast majority of macular diseases can be diagnosed easily. However there are several macular conditions that are not well visualised and therefore optic neuropathy is suspected. They are usually missed because of findings are clinical examination are subtle and

overlooked or the clinical suspicion is not high enough and the correct diagnosis is never considered.

These conditions are:

1) central serous maculopathy
2) cystoid macular edema
3) Diabetic macular ischaemia.
4) Acute macular neuroretinopathy
5) cone dystrophy
6) Toxic maculopathies
7) Idiopathic blind spot enlargement syndrome
8) Choroidal ischaemia.

CLINICAL DISTINCTION BETWEEN OPTIC NEUROPATHY AND MACULOPATHY .

SL.NO	SIGN	OPTIC NEUROPATHY	MACULOPATHY
1	Reduced acuity	Common	Common
2	Dyschromatopsia	Severe	Mild
3	Amsler grid abnormality	Missing portions or gray spots	Distorted or bent lines
4	Afferent pupillary defect	Common	Rare
5	Visual field defects	Central, arcuate, nasal, altitudinal	Central scotoma and midperipheral defects in photoreceptor disease
6	Ophthalmoscopy	Swollen,pale or normal optic nerve	Occasionally pale optic nerve , macular abnormality (pigment atrophy,edema)
7	Photostress recovery	Normal	Abnormal

SL.NO	TESTS	OPTIC NEUROPATHY	MACULOPATHY
1	Electroretinography	Normal	Normal or abnormal (Especially focal ERG)
2	Ocular coherence tomography	Normal	Abnormal
3	Visual evoked response	Large latency delay	Small latency delay

Despite extensive evaluation, it may be impossible to localize the cause of the patient's visual loss . In this setting it is important to consider the possibility of nonorganic or functional vision loss. When doubt exists about the cause of the visual loss, one should consider screening the patient for the treatable causes of optic nerve dysfunction including mass lesions , infections and nutritional processes.

PART II

AIM OF STUDY

The aim of the study is to analyse

- the various causes
- clinical course
- associated findings of patients presenting with temporal pallor of the optic nerve head
- management.

INCLUSION CRITERIA

Patients with temporal pallor of optic disc whose vision showed no improvement with glasses or pin hole.

EXCLUSION CRITERIA

Patients with refractive errors,corneal diseases, cataract and glaucoma were excluded from the study.

MATERIALS AND METHODS

This study was conducted in 50 patients presenting with temporal pallor of the optic nerve head in the Regional institute of ophthalmology, Govt ophthalmic hospital, Chennai during the period of March 2007 to October 2008.

A detailed history of the nature of complaints, history pertaining to smoking, alcohol, diet and long term intake of drugs especially ethambutol was obtained.In all cases visual acuity,detailed anterior segment examination with slitlamp biomicroscopy with special reference to pupillary reflex,posterior segment evaluation with both direct and indirect ophthalmoscope,colour vision and fields were done.X ray skull,CT brain and orbit and MRI brain were taken to detect the cause of temporal pallor.

ANALYSIS AND DISCUSSION

Total number of patients under study-50

AGE INCIDENCE

Table 1

S.L.NO	AGE IN YEARS	NO OF CASES	PERCENTAGE
1	1-10	6	12
2	11-20	5	10
3	21-30	7	14
4	31-40	15	30
5	41-50	12	24
6	>50	5	10
7	TOTAL	50	100

The age incidence showed a preponderance between 3rd to 5^{th} decade.

SEX INCIDENCE

Table 2

S.L.NO	SEX	NO OF CASES	PERCENTAGE
1	Male	35	70
2	Female	15	30
3	Total	50	100

The sex incidence showed a preponderance in the male population.

PRESENTATION OF CASES

Table 3

S.L.NO	COMPLAINTS	NO OF CASES	PERCENTAGE
1	Defective vision	4	8
2	Headache	3	6
3	Defective vision with headache	40	80
4	Non specific	3	6
5	Total	50	100

Figure 9-Age of presentation

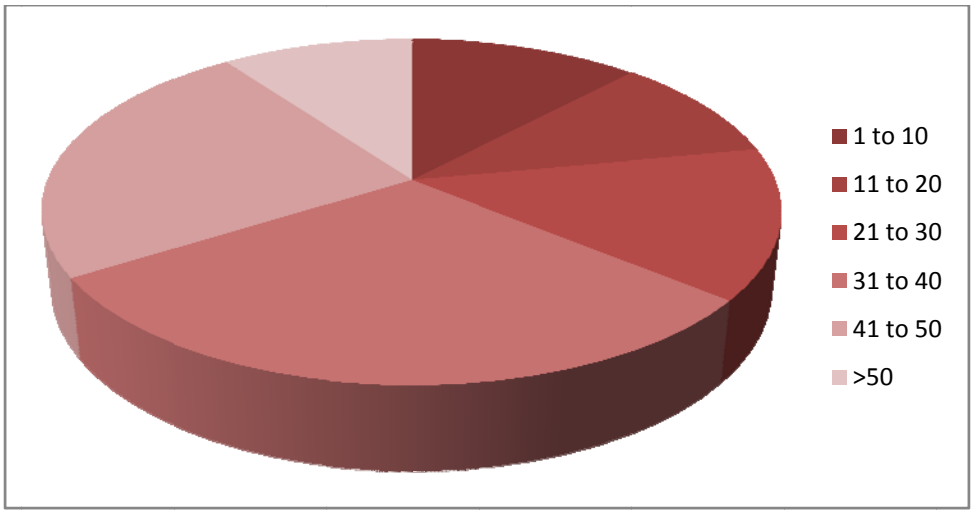
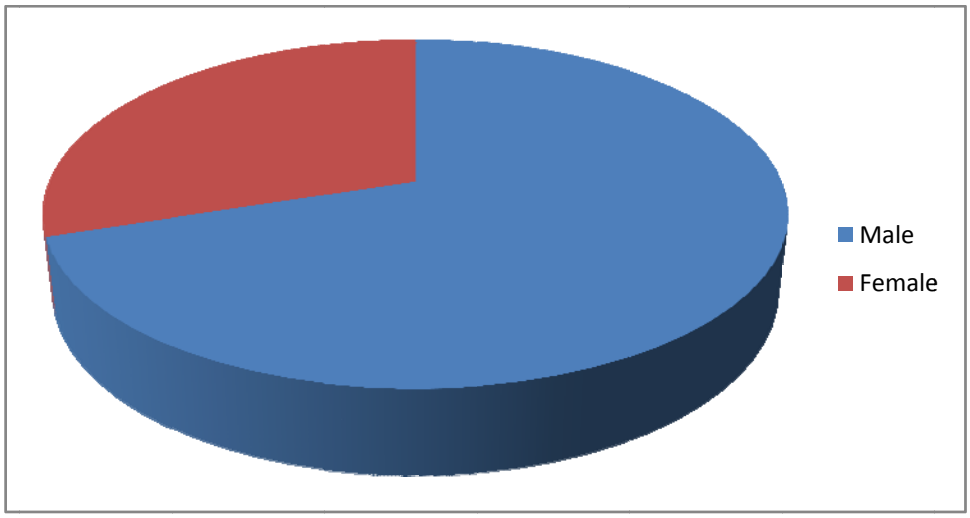


Figure 10-Sex distribution



In my study , 80 % of the patients presented with defective vision and headache. 8% of the patients presented with only defective vision. 6% of patients presented with only headache. 6% of patients presented with nonspecific symptoms like deviation of the eyes.

VISUAL ACUITY

Table 4

S.L.NO	VISUAL ACUITY	NO OF CASES	PERCENTAGE
1	6/6-6/12	8	16
2	6/18-6/24	16	32
3	6/36-6/60	13	26
4	<6/60	12	24
5	Not recordable(child)	1	2
6	Total	50	100

The visual acuity was tested with snellen's chart. In my study, 32% of patients had visual acuity of 6/18-6/24. 26% of the patients had visual acuity of 6/36-6/60. 24% of patients had visual acuity less than 6/60.Though the patients presenting with temporal pallor of the optic disc may have a fairly

Figure 11-Presentation of symptoms

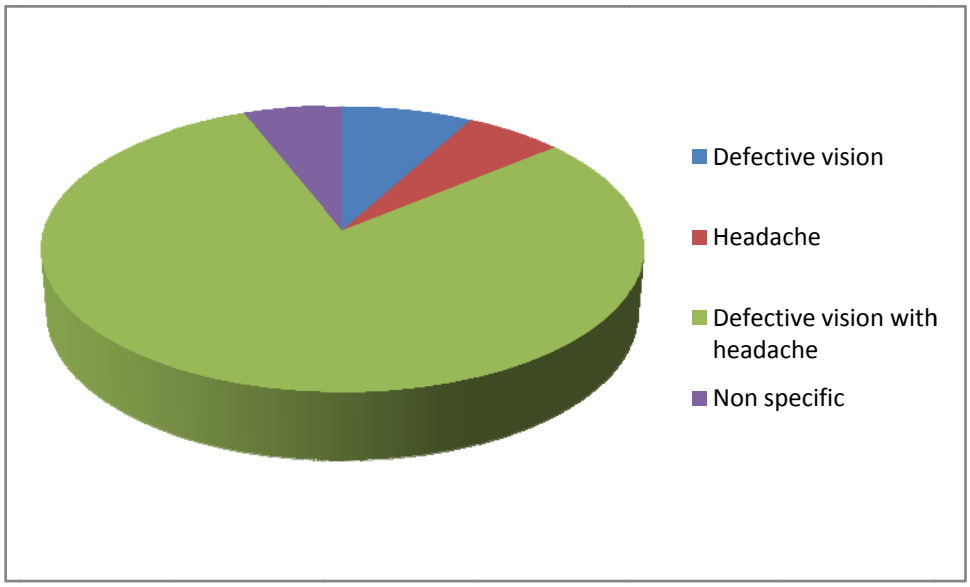
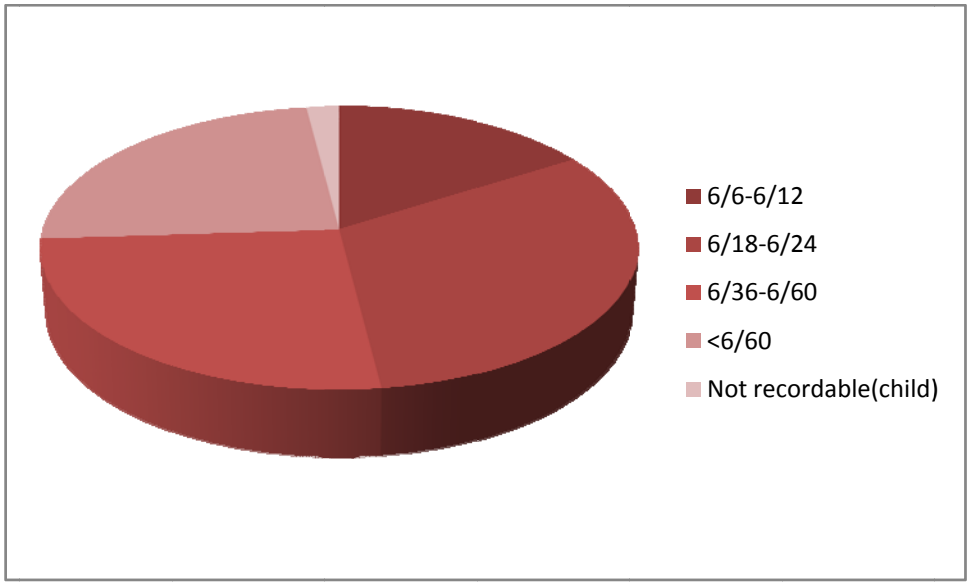


Figure 12-Visual acuity



good vision, it may still be necessary to do necessary investigations to find out the cause of temporal pallor.

ESTIMATION OF COLOUR VISION

Table 5

S.L.NO	COLOUR VISION	NO OF CASES	PERCENTAGE
1	Normal	14	28
2	Defective	30	60
3	Not possible(due to poor vision, children)	6	12
4	Total	50	100

Estimation of colour vision was done with pseudo isochromatic chart. It was defective in 60% of the patients. It was not possible in patients with poor vision and in children in 12% of patients.

FIELDS

Table 6

S.L.NO	FIELD DEFECTS	CASES	PERCENTAGE
1	Normal	20	40
2	Centrocaecal scotoma	9	18
3	Central scotoma	8	16
4	Bitemporal hemianopia	2	4
5	Generalised constriction of fields	4	8
6	Not cooperative(children under 2 years)	1	2
7	Examination not possible(vision<1/60)	6	12
8	Total	50	100

Figure 13-Estimation of colour vision

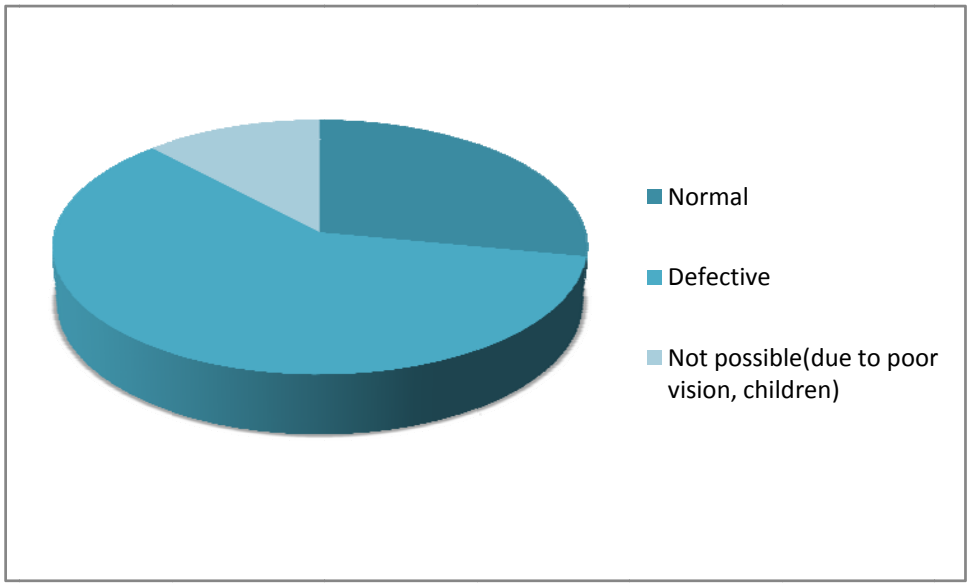
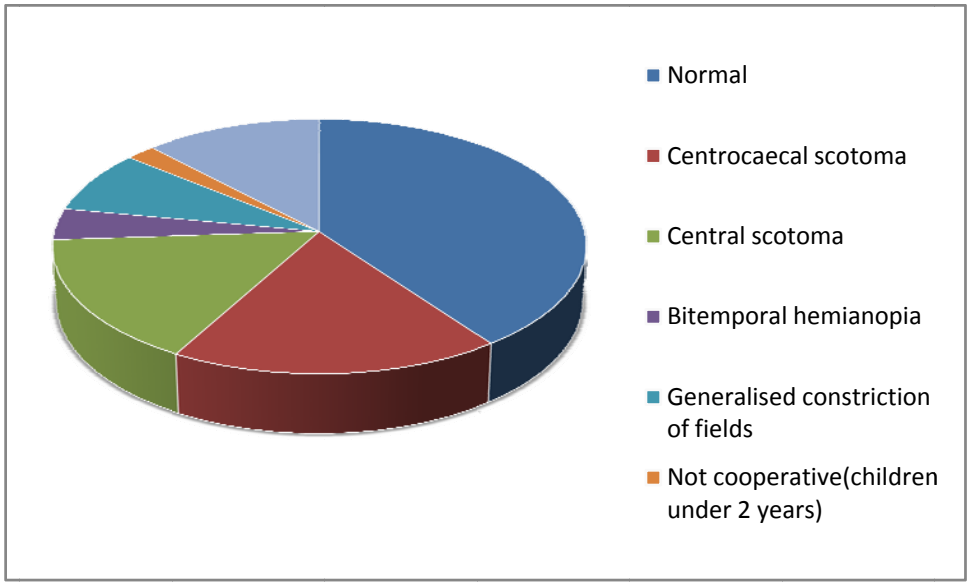


Figure 14-Fields



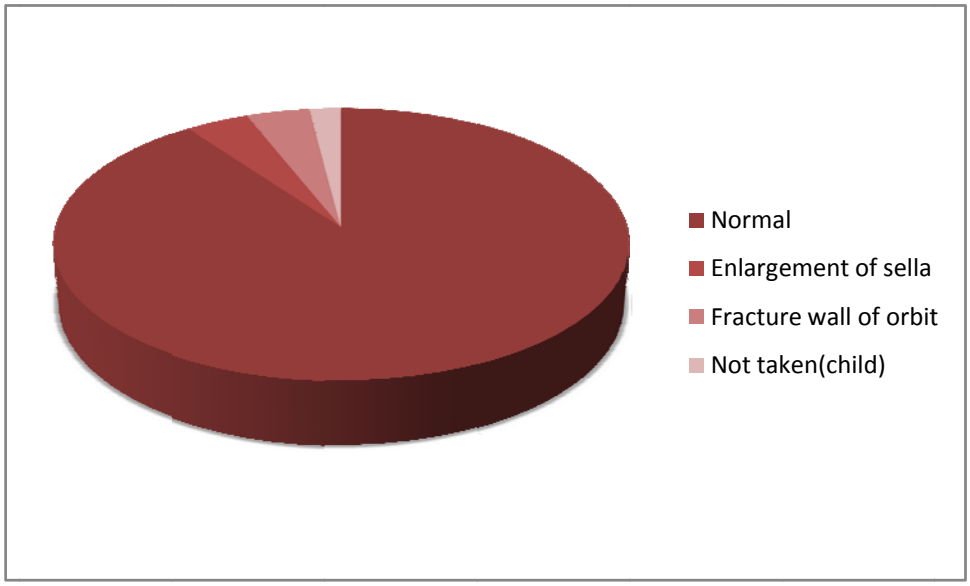
Examination of fields was done with Bjerrum's screen using coloured objects. The fields of the patients who had optic nerve pathology were tested with red targets and those who had macular pathology were tested with blue yellow targets. Patients having tobacco-alcohol amblyopia presented with central and centrocaecal scotoma.Chiasmal compressive lesions presented with bitemporal hemianopia.Patients with retrobulbar neuritis had contraction of the visual fields.Automated perimetry confirmed the findings of the kinetic perimetry.

X RAY FINDINGS

Table 7

S.L.NO	FINDINGS	NO OF CASES	PERCENTAGE
1	Normal	45	90
2	Enlargement of sella	2	4
3	Fracture wall of orbit	2	4
4	Not taken(child)	1	2
5	Total	50	100

Figure 15-X-ray findings



The X-ray findings were normal in 90% of the patients.The enlargement of sella due to pituitary adenoma was found in 4% of the patients. Two out of six patients with traumatic optic neuropathy had fracture in the lateral wall of the orbit.

MANAGEMENT

After appropriate investigations, in 2 patients the findings were suggestive of pituitary tumour and in 3 patients the findings were suggestive of suprasellar meningioma and they were referred to neurosurgery for further management. In the remaining 45 cases, 10 doses of vitamin B1, B6,B12 injections were given on alternate days and neurovitamin supplements were given. All of them were asked to come for regular follow up for one year. Three patients who were on ATT had ethambutol induced toxic amblyopia and were advised to stop ethambutol and to continue the other ATT drugs. Nine patients who had tobacco-alcohol amblyopia were advised to stop taking tobacco and alcohol.

RESULTS

50 cases of temporal pallor of optic disc were taken for study.

In my study,

- 68% of the patients(38 patients) presented between third and fifth decades.
- 70% of the patients(35 patients) were males and 30%(15 patients) were females.
- 80% of the patients(40 patients) presented with defective vision and headache and the visual acuity was not improving with pinhole.
- In 82% of the patients(41 patients), the visual acuity ranged between 6/18 - <6/60.
- In 60% of the patients(30 patients), the colour vision was defective.
- 46% of the patients(23 patients) had field defects. Patients with tobacco-alcohol amblyopia and ethambutol induced toxic amblyopia had central and centrocaecal scotomas(12 patients). Patients with pituitary adenoma had bitemporal hemianopia(2 patients). Patients

with retrobulbar neuritis had generalized constriction of the visual fields(5 patients).

- X-ray findings showed enlargement of sella turcica due to pituitary tumour in 4% of the cases. Out of 6 patients who had traumatic optic neuropathy, the X-ray findings of 2 patients showed fracture in the lateral wall of orbit.

- In 14 % of the patients(7 patients), CT scan findings were suggestive of pituitary tumors, suprasellar meningioma,fracture of the wall of the orbit.

- 18% of the patients(9 patients) who were diagnosed to have tobacco-alcohol amblyopia were advised to stop taking tobacco/alcohol and had slight improvement in vision.

- 6% of the patients(3patients) who were on ATT were advised to stop ethambutol.

In our follow up of the patients for a period of 6 months, patients with tobacco-alcohol and ethambutol induced toxic amblyopia had visual improvement of two lines after stopping the agents and with neurovitamin supplement therapy.

Out of the 50 patients, the causes of temporal pallor were,

Incidental finding	8
Idiopathic	6
Tobacco amblyopia	9
Post traumatic sequelae	6
On ATT(ethambutol)	3
RB Neuritis sequelae	5
Chiasmal compression	2
Suprasellar meningioma	3
Post meningitic sequelae	2
Post lactational	1
Anemia	2
ARMD	2
Motor neuron disease	1

Figure 16-CT brain axial view showing pituitary tumour

Figure 17-CT brain coronal view showing suprasellar meningioma

CONCLUSION

Temporal pallor of the optic disc is one of the common ophthalmic finding in our practice. It could be physiological or pathological.

- Most of the patients with temporal pallor presented between third and fifth decades.
- 2/3 rd of the patients were males who gave history of prolonged intake of tobacco and alcohol.
- Tobacco alcohol amblyopia form the important cause.The other important causes were post traumatic, post neuritic,post lactational, post meningitic, ethambutol induced, compressive lesions and macular diseases.
- It is necessary to emphasise that patients with temporal pallor who have defective visual acuity not improving with glasses, defective colour vision have to be thoroughly investigated to detect the cause of temporal pallor. The commonly encountered field defects were central, centrocaecal scotomos, bitemporal hemianopia and contraction of visual fields which aided in the diagnosis of the etiology.

- Inspite of careful history,clinical examination and detailed investigations the cause of temporal pallor was not detected in 6 patients.

- Regarding the management and prognosis, those presenting early with remediable cause had improvement in visual acuity,(tobacco ,alcohol,nutritional,post neuritic,drugs).Those presenting late like chiasmal compression ,suprasellar meningioma,post traumatic did not show significant improvement.

PartIII

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PROFORMA

1. Serial No
2. OP/IP No
3. Name of the patient
4. Age
5. Sex
6. Address
7. Occupation
8. Complaints Duration
9. History taking:-
    a. History of presenting illness

b. Past history:With reference to optic neuritis,meningitis,pituitary dysfunction,TB,syphilis,diabetes,hypertension

c. Family history

d. Occupational history

e. Socioeconomic history

f. Nutritional history

g. Personal history:With special reference to tobacco,alcohol;their quantity and quality,mode of intake

h. Menstrual history

i. Lactational history

j. Treatment history:With special reference to ATT

10. Clinical examination:-

a. General examination

b. Ophthalmic examination

1. Visual acuity,refraction

2. Anterior segment
3. Pupillary reactions
4. Fundus examination
5. Visual fields(central including red objects and peripheral fields
6. Colour vision
7. Macular function tests

11. Examination of other systems:-

a. CNS

b. CVS

c. RS

d.Abdomen

e. ENT

f. Skin

12. Investigations

a) Complete hemogram

b) Blood glucose profile

c) Urine albumin sugar

d) Mantoux test

e) Blood VDRL

f) X ray skull-AP and lateral view

g) Visually evoked potential

h) CT brain and orbit

13. Diagnosis

14. Treatment

15. Response to treatment

INDEX TO MASTER CHART

A. Name

B. OP No

C. Age

D. Sex-M-Male,F-Female

E. History of tobacco intake

F. History of alcohol intake

G. Other relevant history-Trauma,TBM-Tuberculous meningitis,HIV-Human immuno deficiency virus,GT-Generalised tremors,SHD-Speech and hearing difficulty,CRF-Chronic renal failure

H. Other ocular findings-ACS-Alternating convergent squint,RAPD-Relative afferent pupillary defect,LDS-Left divergent squint,ADS-Alternating divergent squint,PR-SRTL-Pupillary reactions-sluggishly reacting to light,LE-TAPD-left eye-total afferent pupillary defect

I. Visual acuity-NR-Not recordable,RE-Right eye,LE-Left eye,BE-Both eyes,NIP-Not improving with pinhole,NIG-Not improving with glasses

J. Colour vision-NR-Not recordable,N-Normal,D-Defective,NP-Not possible

K. Fields-NR-Not recordable,N-Normal,NP-Not possible,RE-TH-Right eye temporal hemianopia,BTH-Bitemporal hemianopia,CS-Central

scotoma,CCS-Centrocaecal scotoma,GCF-Generalised constriction of fields

L. X ray findings-N-Normal,#LLWO-Fracture left lateral wall of orbit,SE-Sellar enlargement

M.Other investigations-CTO-N-Computerized tomography normal,CTO-#LLWO-CT orbit fracture left lateral wall of orbit,PM-Pituitary mass,N-Normal,PSRT-PL-Photo stress recovery test prolonged,CTB-SSM-CT brain supra sellar meningioma

N. Final impression-RB-Retro bulbar,ATT-Anti tuberculous treatment,ARMD-Age related macular degeneration,MND-Motor neuron disease

MASTER CHART

Prakash	71837	2	M	_	_	_	ACS	NR	NR	NR
Narasimman	81937	6	M	_	_	Trauma	LE RAPD	RE 6/6 ; LE 1/60 NIP	N/NP	N/NP
Kumaran	82340	8	M	_	_	_	_	RE 6/12 ; LE 3/60 NIP	N/D	N
Velan	1402	10	M	_	_	_	LDS	RE 6/6 ; LE 6/18 NIP	N	N
Surya	1440	11	M	_	_	Trauma	LE RAPD	RE 6/6; LE 6/36 NIP	N/D	N
Madhi	831	12	M	_	_	_	ADS	BE 6/24 PH 6/12	N	N
Mani	1726	12	M	_	_	Trauma	LE RAPD	RE 6/6 LE 3/60 NIP	N/D	N
Nagarajan	2159	15	M	_	_	_	_	RE 6/12 PH 6/9 LE 6/18 NIP	N	N
Manivel	2143	21	M	_	_	_	PR- SRTL	RE 6/60 NIP LE NO PL	D	RE TH L NP
Kulandaisamy	2265	29	M	_	_	_	PR- SRTL	BE 6/60 NIP	D	BTH
Manickam	2253	26	M	_	_	Trauma	LE TAPD	RE 6/6 LE 5/60 NIP	N/D	N
Ashok	2229	25	M	_	_	_	RE RAPD LE SRTL	RE 2/60 NIP LE 6/12	D/N	CS/N
Natesan	2046	27	M	_	_	_	_	RE 6/6 LE 6/12 NIP	N	N
Krishnan	3420	31	M	_	_	_	RE RAPD	RE 6/24 NIP LE 6/6	D/N	GCF/N
Alagesan	3498	33	M	_	_	_	RE RAPD	RE 6/36 NIP LE 6/12	D/N	GCF/N
Kamal raj	4176	33	M	_	_	TBM	_	RE 6/18 NIP LE 6/6	D/N	N
Sivanesan	4252	34	M	_	_	_	_	RE 6/12 NIP LE 6/6	N	N
Mahesh	3146	36	M	_	_	HIV	_	RE NO PL LE 6/24 NIP	NP/D	NP/GCI
Vetrivel	3687	36	M	+	+	_	_	RE 6/12 NIP LE 6/18 NIP	D	CCS
Madhialagan	4052	36	M	+	+	_	_	BE 6/24 NIP	D	CCS
Logesh	4906	36	M	+	_	_	_	BE 6/18 NIP	D	CCS
Anandan	5340	37	M	_	+	_	_	BE 6/60 NIG	D	GCF
Chandru	3818	38	M	+	+	_	_	BE 6/36 NIP	D	CCS
Mithran	4959	40	M	+	+	_	_	RE 6/24 NIP LE 6/36 PH 6/24	D	CCS
Kishore	4252	41	M	+	+	_	_	BE 6/36 NIP	D	CCS
Saravanan	7772	41	M	_	_	_	_	RE 6/12 NIP LE 6/9 PH 6/6	N	N
Vijaykumar	8736	43	M	_	_	Trauma	LE RAPD	RE 6/9 LE 1/60 NIP	N/D	N/NP
Thambi		44	M	_	_	_	_	BE 6/12 NIP	D	N
Madhan	9160	45	M	_	_	_	RE RAPD	RE 6/36 NIP LE 6/9 PH 6/6p	D/N	CS/N
Vignesh	10141	47	M	_	_	_	RE RAPD	RE 6/60 NIP LE 6/6	D/N	CS/N
Devaraj	12356	48	M	+	+	_	_	RE 6/36 NIP LE 6/12 NIP	D	CCS
Sampath	12450	48	M	_	_	_	_	RE 6/12 NIP LE 6/24 NIP	D	CCS
Venkatesh	12728	48	M	_	_	_	_	BE 6/12 NIP	D	CS
Mani	12263	52	M	_	_	_	_	BE 6/36 NIP	D	CS
Rajan	13589	59	M	+	+	_	_	BE 6/24 NIP	D	CCS
Vijayalakshmi	13607	17	F	_	_	GT/SHD	N	BE 6/24 PH 6/12	N	N
Vennila	13981	21	F	_	_	_	LE RAPD	RE 6/12 LE NO PL	N/NP	NP
Gajalakshmi	20608	27	F	_	_	_	_	BE 6/36 PH 6/18	N	N
Saroja	24166	4	F	_	_	_	_	BE 6/12 NIP	N	N
Rajeswari	28528	4	F	_	_	_	_	BE 6/18 PH 6/12	N	N
Saraswathi	34829	37	F	_	_	_	_	BE 6/18 PH 6/12	N	N
Nachammal	33189	34	F	_	_	_	_	BE 6/24 NIP	D	N
Panchavarnam	38478	32	F	_	_	CRF	_	BE 6/12 NIP	N	N
Keerthi	44132	38	F	_	_	_	_	BE 6/12 NIP	N	CS
Meena	44446	50	F	_	_	_	RE RAPD	RE NoPL LE 6/18 NIP	NP/N	NP/N
Manohari	50914	51	F	_	_	_	LE RAPD	RE 6/12 LE NO PL	N/NP	N/NP
Malathi	57455	52	F	_	_	_	_	RE 6/24NIP LE 6/12 NIP	N	N
Selvi	60522	53	F	_	_	_	_	BE 6/18 NIP	D	CS

Lakshmi	64533	42	F	_	_	_	LE RAPD	RE 6/24 PH 6/12LE 6/60NIP	N/D	N/GCF
Sivagami	68398	47	F	_	_	_	RE RAPD	RE 5/60NIP LE 6/12	D/N	CS/N

LIST OF SURGERIES PERFORMED

S.No	Name	Age	Sex	IP No	Diagnosis	Date of	Type of surgery
1	Balan	65	M	405952	LE Mature cataract	14.3.07	LE ECCE
2	Mohaideen	55	M	411640	RE Mature cataract	21.3.07	RE ECCE
3	Jayaraman	57	M	412103	RE Immature	4.4.07	RE ECCE
4	Ammu	50	F	412331	RE Mature cataract	18.4.07	RE ECCE
5	Mariammal	55	F	412324	RE Chronic	24.7.07	RE DCT
6	Elumalai	18	M	415623	RE Pterygium	8.5.07	RE Pterygium
7	Sarammal	50	F	423015	LE Chalazion	6.6.07	LE Chalazion
8	Rajammal	58	F	411385	RE Mature cataract	6.7.07	RE ECCE
9	Ponnusamy	55	M	404648	RE Mature cataract	27.7.07	RE SICS
10	Pottiammal	65	F	417340	LE Immature	31.8.07	LE SICS
11	Indirani	52	F	418569	LE Immature	1.10.07	LE SICS
12	Saroja	62	F	418730	RE Immature	6.10.07	RE SICS
13	Venkatalakshmi	48	F	420519	RE Chronic	18.10.07	RE DCT
14	Kuppammal	65	F	419728	RE Mature cataract	5.11.07	RE SICS
15	Ramakrishnan	70	M	420078	RE Nuclear cataract	19.11.07	RE SICS
16	Muniammal	65	F	420835	RE Immature	10.12.07	RE SICS
17	Vasantha	60	F	420412	RE Mature cataract	31.12.07	RE SICS
18	Veerabathran	58	M	421923	RE Immature	31.1.08	RE SICS
19	Kaesavan	52	M	421222	RE Immature	21.2.08	RE SICS
20	Arumugam	65	M	423023	RE Immature	3.3.08	RE SICS
21	Lakshmi	54	F	423815	LE Immature	12.3.08	LE SICS
22	Kowsili	60	F	424725	RE Immature	3.4.08	RE SICS
23	Subramani	60	M	426967	RE Immature	3.6.08	RE SICS
24	Ibrahim	47	M	427510	RE Mature cataract	17.7.08	RE SICS
25	Shanthalakshmi	27	F	429122	RE Divergent squint	25.9.08	RE Squint